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NUTRITION AND VISION – VITAMIN A

One of the oldest diseases known to man is the failure of vision in dim light, commonly known as night blindness. The disease is mentioned in the writings of the ancient Egyptians. In more recent times it has been reported wherever malnutrition has been prevalent—among prisoners, slaves, institution inmates, sailors on long sea voyages, and famine-stricken populations. Night blindness still exists today. In modern civilization where night driving, flying, and many other night activities constitute an integral part of living, the existence of this disorder—now recognized as a specific nutritional disease—becomes a major problem.

Long exposure to bright light has been observed as a concommitant factor in the history of night blindness. There are records, for example, of Brazilian slaves setting out to work in the dim light of early dawn and experiencing no visual difficulties; yet, upon returning at dusk following a day's labors in the sun, there were many complaints of disturbed vision. Recently, night blindness has been reported as being fairly common during the winter in Labrador where the inhabitants practice the ingenious custom of bandaging one eye during the day to preserve it for use at night.

It is interesting to note that the earliest references to this disease in the medical papyri of ancient Egypt some four thousand years ago prescribed a proper corrective treatment—the eating of liver. This treatment is still used today. As described by Wald (1):

> "The Kahun Papyrus, an Egyptian gynecological treatise written about 1850 B.C., contains a long and unpleasant prescription ending with the exhortation 'and make her eat the liver of an ass, raw.' The Ebers Papyrus (ca. 1500 B.C.) prescribed for this purpose the roast liver of an ox, adding that 'By early writers the liver of a black cock was also recommended.' Hippocrates (5th century B.C.) advised 'a raw beef liver (the largest available) steeped in honey'; Pliny the Elder (1st century A.D.) the liver of a goat, since 'it is said that goats can see no less well at night than in the daytime'. During the nineteenth century the liver oil of the cod became the treatment of choice; and still more recently very potent oils and concentrates from the livers of other marine fishes. Not until 1917 was the ingredient of liver oils which cures night-blindness identified with the newly recognized fat-soluble vitamin A."

Thus, from earliest times until a quarter of a century ago, evidence steadily accumulated, culminating finally in the finding that vitamin A in its various

forms is intimately involved in the mechanism of vision. Recognition of this fact came slowly. It was dependent upon two factors. First, the development of the chemistry of vitamin A and of the visual pigments; second, the realization that vitamin A deficiency as manifested in night blindness is a direct expression of a disturbance in the visual mechanism, rather than an indirect reflection of general systemic disorder.

It is now known that vitamin A is essential in the maintenance of the normal epithelium of the eye, as well as of other epithelial tissue. In addition, it plays a direct functional role in vision. A deficiency of this vitamin is the cause of one form of night blindness and of the diseases of the eye known as xerosis, xerophthalmia and keratomalacia. This knowledge has indicated the importance in the diet not only of liver, but of other foods such as milk, cream, butter, green and yellow vegetables, which are rich in vitamin A. This review is concerned with the story of vitamin A as it is involved in the visual mechanism and with the practical significance vitamin A assumes in present-day civilian and military life.

Early Scientific Background:

With the discovery of the fat-soluble vitamin A by McCollum and Davis (2) and Osborne and Mendel (3) in 1913, and its relation to the health of the eye, the way was clear for further investigation of the relationship between vitamin A and the eye. Xerophthalmia and blindness among the children of Denmark during the first World War (4) * was soon traced to lack of vitamin A brought about by the absence of butter in the diet. During the course of these and other investigations, it was noted that night blindness is a frequent symptom preceding or accompanying xerophthalmia. The possible relation between simple night blindness and vitamin A deficiency became evident and many authorities suggested the use of cod liver oil and other vitamin A-rich materials for the treatment of this disorder. Not all cases of night blindness responded to this therapy and it was soon realized that there are several types of night blindness, only one of which is due to vitamin A deficiency. (Other types of night blindness may result from structural eye defects unrelated to vitamin A supply or to deficiency of other nutritional factors.)

As knowledge of the chemistry of vitamin A and related compounds progressed, the significance of these compounds in the visual process slowly unfolded. By 1920 (5) it had already been indicated that vitamin A was closely related chemically to certain types of highly important plant and animal pigments, namely, the carotenoids. Ten years later the relationship between vitamin A and carotene was firmly established by the demonstration that vitamin A is synthesized from beta-carotene in the rat (6). This discovery was rapidly followed by the isolation a year later of vitamin A from fish liver oil, and by the determination of its formula which was shown to be that of half a beta-carotene molecule with water added (7). (Fig. I)

*See *Borden's Review of Nutrition Research*, January 1943.

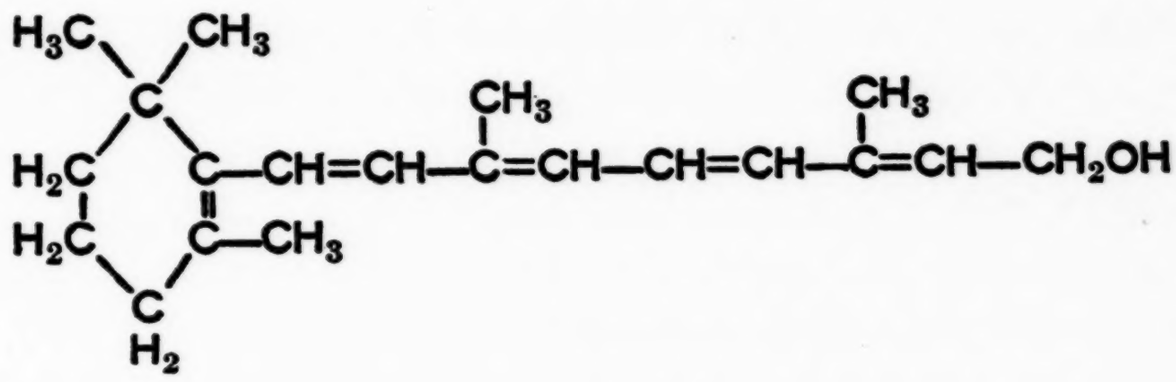

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Fig. 1 Vitamin A (Structural Formula)

Both vitamin A and carotene were found to exert similar physiological responses and the conclusion was reached that carotene is a physiological precursor of vitamin A.

There are three forms of carotene—alpha, beta and gamma. The most effective precursor of vitamin A is beta-carotene which consists of two symmetrical molecules of vitamin A united through the loss of one molecule of water. On hydrolysis beta-carotene yields two molecules of vitamin A. Other provitamins yield, on hydrolysis, only one molecule of vitamin A.

These fundamental researches provided the necessary background for further investigation into the nature of the relationship between vitamin A and vision.

In 1933, Wald (8, 9) identified vitamin A in significant quantities in the retinas and other eye tissues of cattle, sheep, pigs and frogs. In the following year he demonstrated that it participates with a photosensitive eye pigment, rhodopsin, in a complex cycle of chemical reactions in the retina (10, 11, 12). This marked the first recognition of a vitamin in the chemistry of a specific physiological process and led to the development of present theories of visual mechanisms as affected by vitamin A.

The Mechanism of Vision:

The Rods and Cones: Vision is a highly complex process, a great deal of which still remains to be clarified. Certain general principles of vision have been recognized and are, briefly, as follows: The portion of the eye which is sensitive to visual impressions is the retina, an elaborately constructed sense organ in the eye. It is composed of an intricate network of layers of cells of various types. The sensory cells of the retina which respond to stimulation by light waves are the *rods* and *cones,* so called because of their characteristic shapes.

The rod and cone layer is arranged so that the center of the retina is occupied exclusively by cones, while the rest of the retina contains both rods and cones, with rods increasingly predominant toward the periphery.

The Duplicity Theory: Because the retinas of man and of most other vertebrates contain these two different types of sensory cells, a theory has arisen that there are actually two types of vision, namely, *rod-vision* and *cone-vision,* each having different characteristics. This "duplicity" theory postulates that the retina is not a single but a double sense organ. At high light intensities the center of the retina is most effective in vision, whereas at low intensities the peri-

phery is most effective. The duplicity theory associates the cones with vision at high light intensities, and the rods with vision at low light intensities. Further, since color is seen best at high intensities and little or no color is seen at low intensities, the cones are considered as specific receptors for color vision and the rods as general receptors for light regardless of color. (A thorough discussion of the physics and chemistry of vision is given by Hecht (13)).

The change that takes place in the rods and cones is one whereby light energy is converted into nerve impulses. These impulses are carried along the optic nerve to the brain where they are interpreted as a reproduction of the visual pattern initiating the stimulus. The nature of this change is not known. It is believed to be a photochemical process similar to that produced on a sensitized photographic film.

Visual Purple: The basis for the photochemical theory lies in the existence of a rose-colored pigment in the retina. This pigment, known as *rhodopsin* or *visual purple,* is bleached by light and resynthesized in the dark. Its existence and significance were first discovered in 1877 by Franz Boll (14), and its properties studied extensively by Kühne (15). Rhodopsin is found chiefly in the rods, very little, if any, occurring in the cones.

However, since the area of most distinct vision (i.e. the central retina) contains only cones and since many facts support the theory that the initial step in the production of visual inpulses is *the action of light on a photochemical substance,* it was assumed that a photosensitive substance similar to rhodopsin must exist in the cones. This has been proved to be the case by the experiments of Wald (16) on chicken retinas (composed mainly of cones). He extracted from these, two photosensitive substances, one apparently rhodopsin, and the other, a substance exhibiting a maximum absorption at an intensity which corresponds to the maximum sensitivity of the light-adapted eye. The latter substance is believed to belong to the cones and has been named *iodopsin* or *visual violet.*

Visual purple has been identified as a protein containing a carotenoid group. Synthesis of visual purple in the retina is therefore dependent upon the supply of vitamin A, or carotenes, as well as upon the metabolic activity of the retina.

The Visual Cycle: In continuation of his experiments on the occurrence of carotenoids in the eyes of various animals, Wald (17) found that not only is vitamin A a precursor of visual purple, but that it is also a decomposition product of that substance. In other words, vitamin A, which is required for the formation of visual purple, is formed again as the free vitamin when visual purple is decomposed by the action of light. This re-formed vitamin A is then again utilized for the regeneration of more visual purple, and so on, in a continuous cycle. Inasmuch as some vitamin A is lost in each cycle, it is essential that the supply be continuously replenished.

The visual cycle is initiated when light falls upon the retina. Visual purple (rhodopsin) is broken down and bleached to yield a substance known as visual yellow. Visual yellow dissociates into a pigment, retinene and a protein. Some of the visual yellow (retinene-protein) may regenerate a small amount of rhodopsin, but for the most part, retinene is converted into a colorless product which is found to be vitamin A. The newly formed vitamin A then combines

with the remaining protein in the dark to regenerate *visual purple* (rhodopsin). Wald (11) has expressed this schematically as follows:

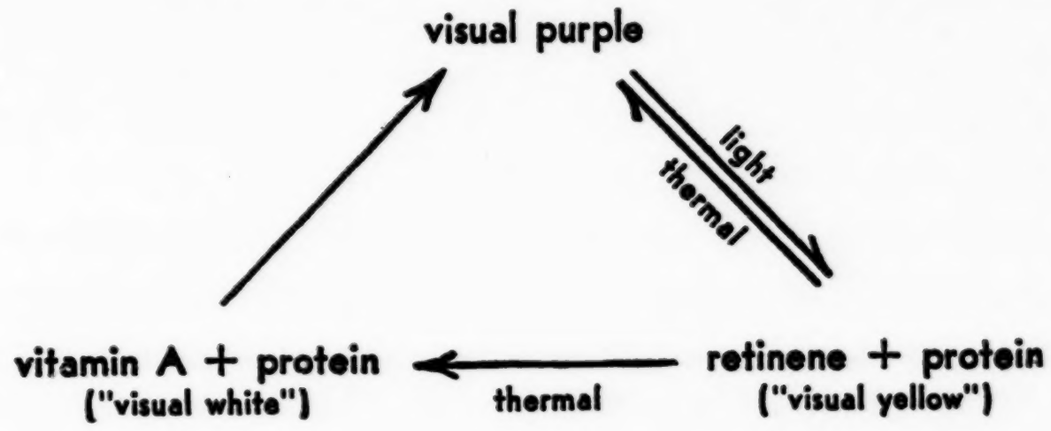

Fig. II Vitamin A in the Visual Cycle

The Interpretation of Night Blindness:

According to the duplicity theory of vision, the rods and cones have different visual functions. The cones are assumed to be responsible for color vision and vision in bright light, while the rods are especially adapted because of their low threshold, to stimulation by light of low intensity and are thus responsible for colorless "twilight vision." Following the discovery of visual purple in the rods (by Franz Boll), it was suggested by Parinaud (18) that twilight vision is dependent upon the presence of visual purple.

Combining the theory of Parinaud concerning the importance of visual purple for twilight vision with the experimental (2, 3) and clinical (4) evidence that vitamin A deficiency leads to xerophthalmia frequently accompanied by night blindness, Fredericia and Holm in Denmark (19) decided to investigate the possibilities of a relation between vitamin A intake and visual purple. Accordingly, they conducted a series of experiments with rats and obtained the following interesting results:

1. The retinas of rats *kept in the dark* showed no difference in the amounts of visual purple, whether the animals were starved for vitamin A or not.
2. The retinas of rats kept in the light long enough to completely bleach the visual purple and then kept in the dark to permit regeneration showed marked differences in the ability to regenerate visual purple, depending upon the vitamin A content of the diet. Vitamin A-starved rats showed a pronounced delay in the regeneration of visual purple.

From these experiments they concluded as follows:

"In human beings a relation between xerophthalmia and a kind of night blindness, often preceding the xerophthalmia, has been suggested by several authors. This kind of night blindness is by some authors thought dependent on a deficiency in the diet of A-vitamin, by others on the exposure of the eyes to intense light. If this kind of night blindness depends on a defect in the function of the visual purple, identical to that observed in rats starved for A-vitamin, both views contain part of the truth."

These experiments indicated at last that there was indeed an important relation between vitamin A intake, the ability to regenerate visual purple, and, as demonstrated in a succeeding experiment (20), the development of night blindness and difficulty in adaptation of the eyes in the dark. Similar experi-

ments by Tansley (21) several years later confirmed the work of Fredericia and Holm.

It was not until eight years later when Wald (8, 9) discovered significant amounts of vitamin A in the retina, and later showed it to be an integral part of visual purple, that the complete explanation of night blindness and faulty dark adaptation was forthcoming.*

In summary then, the phenomenon of night blindness falls in very well with the duplicity theory of vision. In night blindness, vision is subnormal in dim light and the eye does not exhibit its normal ability to adapt or accustom itself to darkness. According to the duplicity theory, the rods are responsible for twilight vision. Since stimulation of the rods is dependent upon a photochemical change involving visual purple, it is apparent that a lack of vitamin A, which constitutes an integral part of visual purple, is responsible for faulty night vision.

Diagnostic Aids In Detecting Vitamin A Deficiency:

Night blindness varies from mild states detectable only by instrumental means to severe disability. In the mild forms the subject is unaware of the defect. In continued vitamin A deficiency, the disorder soon progresses to difficulty in reading or sewing at night, or finding a seat in a darkened theater; stumbling in the dark; trouble in night driving, such as increased sensitivity to approaching headlights, failure to see turns, road obstructions or pedestrians. Severe vitamin A deficiency leads to progressively worse eye conditions such as xerosis and xerophthalmia which are characterized by actual degenerative structural changes in the eye (24).

Photometers: Following the establishment of vitamin A as a factor in the visual cycle, it seemed logical to use visual response for the detection of early stages of vitamin A deficiency.

A number of such tests have been devised for this purpose. One of the earliest of these was developed by Edmund in Denmark (25). In this procedure the discrimination of light intensities by the subject serves as an index of visual function. In this country, however, the generally accepted method for detecting impaired vision is the so-called dark adaptation test. The general principle of this test is to measure the amount of light and time required for perception in the dark following exposure to bright light. Those with normal dark adaptation (an adequate regeneration of visual purple) require less light and less time after bleaching than do deficient subjects. This difference can be measured quantitatively. The use of such delicate methods of measurement serves to indicate early or mild deficiencies of vitamin A which may otherwise be undetectable.

*It is interesting to note that in this eight year interval some glimmerings of the truth were becoming evident through the continued experiments of Holm (22) and of Smith *et al* (23) in which xerophthalmia in rats was successfully cured by the feeding of fresh calf retinas or dried pig retinas. Wald (8), in similar experiments, analyzed the retina extracts for vitamin A potency, thereby confirming the fact that their curative properties were due to the presence of vitamin A in the retinas.

According to Jeghers (26), using the biophotometer, photometric evidence of night blindness could be detected six days after the production of a true vitamin A deficiency in an experimental subject. It was not until the deficiency had lasted for five weeks that the patient could detect any signs of a disturbance in vision. Hecht and Mandelbaum (27), using the Hecht-Schlaer adaptometer, detected signs of dietary avitaminosis as early as one day following the absence of vitamin A from the diet.

Several instruments, as indicated above, are in use for the measurement of dark adaptation. They have not yet been so perfected as to eliminate certain sources of error due to methods of testing and to the subjective responses of the patient. Nevertheless, tests made with these instruments under properly controlled conditions and with effective critical judgment of the operator give reasonably reliable results for average clinical work and serve, therefore, as valuable diagnostic aids in the detection of early vitamin A deficiencies.

Other effective means of detecting early vitamin A deficiency are: (1) The biomicroscope which, as its name implies, is a microscope especially adapted to examination of living eye tissue (28); (2) Determination of the vitamin A and carotene content of the blood.

Clinical Studies:

Prior to about 1930, vitamin A deficiency was not recognized until night blindness, xerophthalmia or keratomalacia developed. All of these constitute manifestations of severe and well advanced stages of vitamin A deficiency and are rare compared to the milder, subclinical degrees of deficiency. Some of the first studies in the United States designed to detect early signs of vitamin A deficiency by photometric means were those of Jeans and co-workers in Iowa (29, 30, 31). Using the biophotometer, they tested 213 children from the outpatients clinic at the Children's Hospital in Iowa City. It was found (29) that forty-five of these children exhibited subnormal dark adaptation. Of these, approximately half (twenty-one) were kept under observation and given a good diet including cod liver oil. These children all regained normal dark adaptation in an average of twelve days.

In a later survey of Iowa school children (30), Jeans *et al* found 26% of a rural group and 53% of a village group to have abnormal dark adaptation; in an urban group they found that 56% of children from families on a high economic level, 63% of children from families on a middle level and 79% of children from low income groups exhibited abnormal dark adaptation. Of a total of 78 village and rural children deficient in vitamin A, who continued under observation and treatment with vitamin A, all but three regained normal dark adaptation.

The work of Jeans *et al* on children was later supplemented by that of Park (32, 33) and Jeghers (26, 34) on adults. Jeghers found (34), in a study of 274 adults, that a mild vitamin A deficiency is fairly common among many

supposedly healthy persons. Factors leading to such deficiency were financial status, peculiarities in choice of foods, ignorance of proper diet and attempts to reduce weight by limiting the diet. It was further found that vitamin A deficiency may result, despite an adequate intake of the vitamin, under conditions which " (a) increase the metabolic need for vitamin A (fever, infection, elevated basal metabolic rate, rapid growth and pregnancy); (b) interfere with the proper absorption from the gastro-intestinal tract; or (c) interfere with the conversion of carotene or storage of vitamin A in the liver."

In contrast to these studies, Lewis and Haig (35) reported in 1940 that vitamin A deficiency among children (studied in New York City) is an uncommon disorder. Of 144 children, all of whom were hospital patients, only one was shown to have abnormal dark adaptation. This child, because of abdominal pain, was on a restricted diet which was low in vitamin A. Administration of a massive dose of vitamin A (120,000 units) restored dark adaptation to normal in thirty-five minutes. The effect of this dose lasted twenty days and was not regained until another dose was given. Placing the child on an adequate diet maintained the normal eye function.

Lewis and Haig attribute the difference in results to variations in reliability of instruments and methods used, pointing out that the statistical error involved in the biophotometer is high, whereas greater accuracy is believed to be possible with the adaptometer. A critical survey of this problem has been given by Hecht and Mandelbaum (25).

Practical Implications:

Jeghers (36, 26) has discussed in detail the vital importance of night blindness caused by vitamin A deficiency, as it is related to traffic problems. In a period of about eight months, he found twenty-three cases of automobile accidents due to night blindness. The drivers in these cases reported that they were easily dazzled and blinded by headlights from other automobiles and that they saw the road, road edge, markers and pedestrians only with difficulty. Vitamin A therapy, in all cases, restored normal dark adaptation and eliminated night blindness.

Statistics indicate that the motor vehicle accident rate on a mileage basis is much higher at night than in the daytime. The National Safety Council (37) has shown, for example, from data collected in a typical eastern state in 1933, that accidents and fatalities due to automobiles reached a peak at dusk and for the night hours maintained a high level in spite of the fact that the traffic rate declined rapidly. Jeghers suggests that much of this may be attributed to poor dark adaptation.

The importance of vitamin A for normal vision in night flying has been recognized by military authorities. Jeghers recommends that normal dark adaptation be incorporated as a requirement in the physical standards for aviators and that dark adaptation tests be included in routine examinations for drivers' licenses.

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The present war has stimulated investigation along these lines and in almost all countries now engaged in combat it is the practice to feed foods rich in vitamins (especially vitamin A) as well as vitamin supplements to aviators.*

Vitamin A Requirements:

Minimum vitamin A requirements have been set at 4000 U.S.P. units daily for adults (Food and Drug Administration), while the recommended daily allowance is 5000 U.S.P. units (National Research Council). Pregnancy and lactation increase the vitamin A requirement, the recommended daily allowance being 6000 U.S.P. units for the formerand 8000 for the latter.

The recommended daily allowances for children (ages 1-12) range from 2000 to 4500 U.S.P. units per day; for adolescents, (13-20) from 5000 to 6000 units per day.

Nutritionists state that these requirements can be met by the inclusion in the diet of readily available common foods which provide adequate amounts of vitamin A or its precursors. Cow's milk, human milk, eggs, cheese, butter, fortified margarines, cream, ice cream, liver, and cod liver oil are all good sources of vitamin A. Spinach, watercress, carrots, green peas, lettuce, oranges and tomatoes provide additional good sources.

Conclusion:

In addition to its role in the maintenance of normal epithelial tissue, vitamin A plays a functional role in the chemistry of vision.

The visual process is initiated by the action of light upon visual purple (rhodopsin), a vitamin A-protein pigment in the retina. This action destroys visual purple by converting it to visual yellow. In the regeneration of visual purple from visual yellow, some vitamin A is lost. This necessitates continuous replenishment of the supply of the vitamin. Lack of sufficient vitamin A hinders the regeneration of visual purple, thereby resulting in loss of visual acuity, or the ability to see well in subdued light. This condition is known as night blindness and is one of the earliest symptoms of vitamin A deficiency in man.

The detection of this visual disturbance by photometric methods has served as a practical means of uncovering early, subclinical deficiencies of vitamin A.

The relation of vitamin A to the visual cycle is of more than academic or scientific interest. Its importance in night driving, in night flying and in general vision in subdued light, especially under war time conditions (dim-out, black-out, etc.), are strong and cogent reasons for the inclusion of optimum amounts of this vitamin in the diet.

*Very recently, it was discovered by Squadron Leader McCrae and Wing Commander Lyle of the Royal Air Force in Malta that a new, unidentified vitamin in citrus juices is also effective in curing night blindness. The new vitamin is neither vitamin A or C; it may be vitamin P, but this has not yet been determined. Its mode of action is also not yet clear. It is being used successfully, in combination with vitamin A, to improve night vision of British fliers (38).

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NEWS DIGEST

WHOLE MILK POWDER having excellent keeping qualities may result in the near future from recent research into factors causing deterioration in flavor quality of this product. It was formerly thought that the development of so-called oxidized flavor in fluid milk was due to the oxidation of the butterfat. Oxidized flavor has now been shown definitely to be due mainly to the oxidation of the lecithin of the fat globule membrane rather than to the oxidation of the butterfat alone.

On the assumption that lecithin may be a factor in the keeping quality of dried whole milk as well, whole milk powder samples were prepared from a fluid milk of considerably reduced lecithin content. It was found that those samples containing the least lecithin had the best keeping quality over a test period of several months.

—1201

HONEY has been found to contain valuable vitamins and minerals. Notable among the minerals present are iron, copper, sodium, potassium, manganese, calcium, magnesium and phosphorus. The vitamins present are primarily vitamin C and the B complex vitamins. Improved assay techniques have made possible more precise analysis of the B vitamins in honey than heretofore. A recent report supplying such analytical data indicates that considerable variations occur in different honey specimens, due to the source, age and pollen content of the honey. Representative vitamin values for a group of new honeys are reported as follows:

	Micrograms per 100 grams
Riboflavin	26.3
Pantothenic acid	54.4
Nicotinic acid	108.5
Thiamin	4.4
Pyridoxine	10.0

—1202

THIAMIN CONTENT OF PORK has been considerably increased by including greater amounts of thiamin in the diet of the pig. A report issued a few months ago describes an experiment wherein three lots of pigs were fed rations containing 5716, 3447 and 1318 micrograms of thiamin per pound of feed or approximately 29, 17, and 7 milligrams of thiamin respectively, per animal per day. No changes in growth rate or feed consumption were apparent despite the variations in thiamin intake.

Analysis of meat from these animals, however, revealed considerable variations in thiamin content. An increase of about 100% in thiamin content of pork muscle resulted when the thiamin intake was increased from 1318 to 3447 micrograms per pound of feed. A further gain of from 15 to 20% resulted from an increase in the thiamin content of the feed from 3447 to 5761 micrograms per pound.

Average Thiamin Values (Fresh Basis)

	Low level of thiamin intake Micrograms/gram	High level of thiamin intake Micrograms/gram
Pork shoulder	7.9	17.3
Center loin	9.5	23.1
Ham end of loin	10.3	23.9

Limited observations with two pigs seem to indicate that pork muscle tissues almost reach the approximate saturation point, as far as thiamin storage is concerned, when the thiamin content of the ration is about 5800 micrograms per pound.

—1203

CHEESE, long accepted as a food of high nutritional value, now assumes increased importance in the food economy. Experimental investigations reveal that cheese is an excellent source of highly digestible protein and is high in energy value because of its high fat content. It has been found that Canadian Cheddar cheese, for example, retains a considerable portion of the nutrients originally present in the milk; namely, 60 percent of the calcium, 50 percent of the phosphorus and 20 percent of the riboflavin. Most of the lactose and thiamin of the milk used, however, are lost in the preparation of the cheese.

—1204

CALCIUM ASCORBATE is a newly synthesized ascorbate which promises to be of great therapeutic value in war surgery. It has been found that the calcium-vitamin C compound is highly effective in bone healing. Calcium has long been known as an essential nutrient in bone development and vitamin C has been demonstrated to speed the healing of wounds in cases of undersaturation in the vitamin. The combination of the two in the form of calcium ascorbate has been found to be more effective in the healing of bone fractures than either calcium gluconate or vitamin C alone, the latter two having been used for some time with moderate success.

—1205

For further information or additional copies address: R. Woods, *Editor,* Borden's Review of Nutrition Research, 350 Madison Avenue, New York 17, N. Y.

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